International Journal of Obesity (1990) **14**, 515–525
Received 10 July 1989; accepted 1 December 1989

THE RELATIONSHIP OF BODY FAT DISTRIBUTION TO BLOOD PRESSURE IN NORMOTENSIVE MEN: THE NORMATIVE AGING STUDY

Rebecca J. TROISI[1,3], Scott T. WEISS[3], Mark R. SEGAL[3], Patricia A. CASSANO[3], Pantel S. VOKONAS[2] and Lewis LANDSBERG[4]

[1]*Normative Aging Study, VA Outpatient Clinic and* [2]*Section of Preventive Medicine and Epidemiology and the Evans Memorial Department of Clinical Research, Department of Medicine, University Hospital, Boston University School of Medicine, Boston, MA;* [3]*Channing Laboratory, Department of Medicine, Harvard Medical School and Brigham and Women's Hospital; and* [4]*Charles A. Dana Research Institute, Beth Israel Hospital and* Joint Department of Medicine, Harvard Medical School, MA, USA.

Body fat distribution may be a more specific marker than obesity for risk of cardiovascular disease and diabetes. The relationship between body fat distribution and sitting systolic and diastolic blood pressure was examined in a cross-sectional analysis of 1936 normotensive men aged 21 to 80 years. In this analysis body fat distribution was represented by the ratio of abdomen circumference to hip breadth (denoted as WHbR). Pearson product-moment correlations adjusted for age revealed a positive correlation between WHbR and both systolic and diastolic blood pressure ($r = 0.13$ and $r = 0.14$, respectively). In a multiple linear regression model controlling for age, smoking status and body mass index (BMI), WHbR was associated with systolic blood pressure [regression coefficient (standard error) = 3.58 (1.8), $P = 0.048$)], but had much less of an association with diastolic blood pressure [regression coefficient (standard error) = 1.90 (1.3), $P = 0.141$]. Further adjustment for alcohol intake decreased the association between WHbR and systolic blood pressure [regression coefficient (standard error) = 2.90 (1.81), $P = 0.110$]. Body fat distribution, as represented by WHbR was associated with level of systolic blood pressure independently of overall level of obesity (BMI) in normotensive men; adjustment for alcohol intake attenuated the relationship. These data suggest that dietary factors, notably alcohol intake, may influence the effect of body fat distribution on blood pressure.

Keywords: body composition, blood pressure, alcohol drinking.

Introduction

Fat patterning may be a more specific marker of cardiovascular disease and diabetes risk than total fat or obesity. Vague[1] reported an increased risk of diabetes in persons with a centripetal accumulation of body fat and subsequent studies have confirmed this association[2–7]. Studies using alternate measures of centripetal and peripheral fat to represent body fat distribution report similar findings with blood pressure. Subscapular skinfold thickness, a measure of centripetal fat, was significantly associated with systolic and diastolic blood pressure[8] and with an increased incidence of hypertension[9] independently of

Correspondence to: Scott T. Weiss, Channing Laboratory, 180 Longwood Ave., Boston, MA 02115, USA.

triceps skinfold thickness, a measure of peripheral fat. The ratio of subscapular to lateral calf skinfold, another index that contrasts centripetal with peripheral fat, has also been associated with systolic blood pressure[10]. More recently, the ratio of waist to hip circumference has become popular as an index of fat distribution[2,11–14]. An increased incidence of hypertension[11,13], stroke and ischemic heart disease[14] has been associated with an increased ratio of waist to hip circumference. Blood pressure has also been shown to have a strong positive correlation with the ratio of waist-to-hip circumference[2,11,12].

The association between measures of centripetal fat accumulation and blood pressure is independent of body mass index[2,11,12], and age[2,10,11] and has been demonstrated in both men[8,10–12] and women[2,8,11]. However, the role played by other factors, including cigarette smoking and the dietary intake of alcohol is not well studied. Although many studies have documented a positive relationship between alcohol intake and blood pressure[15–22], few have examined in detail the multivariate relationships among alcohol, body fat distribution and blood pressure.

In the current study the relationship between body fat distribution and blood pressure was assessed in a normotensive, non-obese male population. Cross-sectional data unselected for body composition were available from the Normative Aging Study (NAS).

Methods

The Normative Aging Study is an ongoing longitudinal, multidisciplinary study established by the Veterans Administration in 1961. The NAS population is 98 percent white and is higher in socioeconomic characteristics than the general local population in Boston, MA. Details of the study protocol have been presented elsewhere[23]. Volunteers were screened based on clinical, laboratory, radiologic, and electrocardiographic criteria to provide an initially healthy population. History or presence of coronary heart disease, diabetes, cancer, peptic ulcer, gout, recurrent asthma or bronchitis were criteria for exclusion from the study. Subjects were also excluded if their systolic blood pressure exceeded 140 mm Hg or their diastolic blood pressure exceeded 90 mm Hg (although less than 5 percent, $n = 123$, of the cohort had values that exceeded the blood pressure criterion). Body composition and hyperlipidemia were not screening criteria.

Blood pressure was taken with a standard mercury sphygmomanometer and a 14 cm cuff. Systolic and fifth phase diastolic blood pressure were measured to the nearest 2 mm Hg in each arm and averaged to provide one systolic and one diastolic measurement. Information on cigarette smoking status was collected by interview. The participants were categorized as never, current or former smokers; to be considered a former smoker the subject must have refrained from smoking for at least one year. In the statistical analyses, current smokers were compared to never-smokers and former smokers were compared to never-smokers. Alcohol consumption was a dichotomous variable based on the response to the following question from the Cornell Medical Index: 'do you usually take two or more drinks per day?'[24]

The analyses used data from the baseline physical examination of the Normative Aging Study cohort conducted between 1962 and 1971. A total of 2280 men aged 21–82 years, with a mean age of 42, were accepted into the study. Of these 2280 subjects complete data were available for 2059 (90 percent). The majority of missing observations were a result of subjects failing to return for an anthropometric examination. The 213 men without anthropometric data had mean blood pressure values that were similar to those for the final study group, but they were on average 2 years younger than the final study group (mean age ± s.d. = 39.54 ± 9.43 years versus 42.29 ± 9.44 years). The other eight subjects were missing at least one of the variables included in the analyses performed in this investigation. One hundred and twenty-three more subjects were excluded based on systolic blood pressure values over 140 mm Hg or diastolic blood pressure values over 90. The 123 subjects excluded on blood pressure were significantly older (mean age ± s.d. =47.87 ± 11.47 versus 41.93 ± 9.18), slightly heavier (mean BMI ± s.d. = 26.87 ± 3.06 versus 25.69 ± 2.8) and had a greater WHbR (mean WHbR ± s.d. = 2.63 ± 0.18 versus 2.54 ± 0.16) compared to the study sample. There was no difference in alcohol intake between the subjects excluded on blood pressure and the study sample. All of the statistical analyses used the 1936 normotensive subjects with complete data.

A series of anthropometric measurements were made on each participant with the subject standing erect with his feet together (except for the hip breadth measurement). Measurements were taken with the subject in undershorts and socks only. Anthropometric measurements included: height measured against a wall chart to the nearest 0.1 inch; weight measured on a balance beam scale to the nearest 0.5 pound; hip breadth measured without exerting pressure at the widest breadth of the hips in a sitting position with the anthropometer (Gneupel, GPM, Switzerland) on the greater trochanter; abdomen circumference measured at the level of the umbilicus. Body mass index (BMI) was calculated as weight (kg) divided by height (m) squared. The ratio of abdomen circumference to hip breadth was formed (denoted as WHbR). The coefficients of variation for abdomen circumference and hip breadth were 8.7 and 6.3, respectively, based on data from the baseline anthropometric examination[25].

The study variables were examined for skewness and kurtosis of distribution. Transformations to improve the linearity assumption were not needed. Scatterplots and Pearson product-moment correlations were used to assess the degree of association among the independent variables and between the independent variables and the measures of blood pressure. The mean values of the independent and outcome variables were compared using a general linear model between alcohol intake groups after adjustment for the effects of age. Multiple linear regression was used to assess the relationship between blood pressure and WHbR while controlling for age, cigarette smoking, BMI, and alcohol intake. Interaction terms for alcohol intake and smoking, and WHbR and BMI were included in a second set of regressions.

All statistical analyses were performed using Statistical Analysis System (SAS) AOS/VS version 5.04[26]. *P*-values less than 0.05 were considered statistically significant.

Results

Table 1 presents the descriptive statistics of the study population. Twelve percent of the participants reported consuming two or more drinks per day within the previous year. Smoking and drinking were related behaviors; among subjects who reported drinking two or more drinks per day 53 percent (124) were current smokers, 32 percent (75) were former smokers and 15 percent (35) were never-smokers.

The correlations among study variables are presented in Table 2. BMI was highly correlated with WHbR ($r = 0.53$, $P < 0.0001$). Age was also positively related to WHbR ($r = 0.26$, $P < 0.0001$). Statistically significant correlations between WHbR and sitting systolic and diastolic blood pressure were demonstrated ($r = 0.13$ and 0.14, respectively, $P = 0.0001$) after adjustment for age. The correlations between BMI and blood pressure were of about the same magnitude (systolic $r = 0.15$, and diastolic $r = 0.21$, $P = 0.0001$).

To determine if alcohol use affected blood pressure levels and body fat distribution, means for blood pressure and WHbR were examined by level of alcohol intake (Table 3) after adjustment for age. Differences in blood pressure levels between drinking groups were slight but statistically significant. The mean systolic blood pressure for men consuming two or more drinks per day was 2.44 mm Hg greater than for men drinking less ($F = 11.94$, $P = 0.0006$). Similarly, the mean diastolic blood pressure was 1.30 mm Hg greater for men drinking two or more drinks per day compared to men drinking less ($F = 6.57$, P

Table 1. *Descriptive statistics for the study variables (n = 1936).*

	Mean	*s.d.*
Age	41.93	9.18
Height (cm)	175.55	6.48
Weight (kg)	79.26	10.35
BMI [wt(kg)/ht(m)^2]	25.69	2.81
Abdomen circumference (cm)	92.97	8.19
Hip breadth (cm)	36.55	2.28
WHbR	2.54	0.16
Systolic BP (mm Hg)	122.31	10.21
Diastolic BP (mm Hg)	76.30	7.35
	n	%
Cigarette Smoking		
Current	778	40.2
Never	556	28.7
Former	602	31.1
Alcohol intake		
< 2 drinks/day	1702	87.9
≥ 2 drinks/day	234	12.1

Table 2. *Matrix of correlations among blood pressure, body composition and age.*

	Systolic BP	*Diastolic BP*	*WHbR*[a]	*BMI*[a]	*Age*
Systolic BP	1.00	0.61*	0.13*	0.15*	0.11*
Diastolic BP		1.00	0.14*	0.21*	0.10*
WHbR			1.00	0.53*	0.26*
BMI				1.00	0.03
Age					1.00

* $P = 0.0001$. [a] Correlations among BMI, WHbR, systolic and diastolic blood pressure are adjusted for age.

Table 3. *Means of study variables by level of alcohol intake adjusted for age.*

	Alcohol intake < 2 drinks/day (n = 1702)	≥ 2 drinks/day (n = 234)	*P*
BMI	25.71	25.52	0.3520
WHbR	2.54	2.57	0.0005
Systolic BP	122.01	124.45	0.0006
Diastolic BP	76.14	77.44	0.0105

= 0.0105). Subjects drinking two or more drinks per day had a greater mean WHbR compared to subjects drinking less (F = 12.05, $P = 0.0005$). Body mass index and age were not significantly different between levels of alcohol intake. Multiple linear regression analysis was performed to examine whether the association between alcohol intake and WHbR was confounded by an association with other variables (Table 4). The positive association between alcohol intake and WHbR remained significant after adjustment for BMI, age, and smoking. When alcohol intake was added to the regression model the R^2 increased from 0.3460 to 0.3525; alcohol intake added an additional 1.8 percent of the total variance in WHbR explained after controlling for other factors.

Regression analyses were performed to assess the relationship between WHbR and blood pressure with and without alcohol intake in the model. After controlling for age, smoking status and BMI (Tables 5 and 6), WHbR was associated with systolic blood pressure, but the association with diastolic blood pressure was not statistically significant. When alcohol intake was added to the model, the association of WHbR with systolic blood pressure was no longer statistically significant and the unstandardized regression coefficient for WHbR decreased from 3.58 to 2.90 (Table 5). In the regression on diastolic blood pressure the unstandardized regression coefficient decreased from 1.90 to 1.49 when alcohol was included in the model (Table 6).

The regression coefficients for age and BMI remained relatively stable across the models. Age and BMI were significantly and positively associated with both systolic and diastolic blood pressure. The regression coefficient for former smoking decreased 23 percent and the regression coefficient for current smoking increased 15 percent with the addition of alcohol intake to the model. Current smoking was negatively associated with systolic and diastolic blood pressure.

Table 4. *Parameter estimates from a multiple linear regression model with WHbR as the dependent variable (final $R^2 = 0.35$).*

Independent variables	β	*s.e.*(β)	*P*
BMI	0.029	0.001	0.0001
Age	0.004	0.000	0.0001
Current	0.021	0.007	0.0039
Former	0.013	0.007	0.0872
Alcohol	0.039	0.009	0.0001

Table 5. *Parameter estimates from multiple linear regression with systolic blood pressure as the dependent variable; with and without adjustment for alcohol intake.*

	Unadjusted for alcohol ($R^2 = 0.04$)				*Adjusted for alcohol ($R^2 = 0.05$)*			
Independent variables	β	*s.e.*(β)	*P*	*R^2 on entry*[a]	β	*s.e.*(β)	*P*	*R^2 on entry*[a]
Age	0.080	0.026	0.0025	0.0046	0.083	0.026	0.0018	0.0048
BMI	0.380	0.096	0.0001	0.0076	0.404	0.096	0.0001	0.0086
Current *v.* never	−1.608	0.573	0.0051	0.0039	−1.848	0.574	0.0013	0.0051
Former *v.* never	0.663	0.589	0.2604	0.0006	0.509	0.589	0.3875	0.0003
WHbR	3.579	1.809	0.0480	0.0019	2.900	1.811	0.1096	0.0012
Alcohol	—	—	—	—	2.658	0.703	0.0002	0.0070

[a] The increase in the R^2 associated with the addition of each independent variable while controlling for the other independent variables.

Table 6. *Parameter estimates from multiple linear regression with diastolic blood pressure as the dependent variable; with and without adjustment for alcohol intake.*

Independent variables	*Unadjusted for alcohol ($R^2 = 0.06$)* β	s.e.(β)	P	R^2 on entry[a]	*Adjusted for alcohol ($R^2 = 0.07$)* β	s.e.(β)	P	R^2 on entry[a]
Age	0.044	0.019	0.0192	0.0027	0.045	0.018	0.0155	0.0028
BMI	0.409	0.069	0.0001	0.0171	0.423	0.069	0.0001	0.0182
Current *v.* never	−1.857	0.408	0.0001	0.0101	−2.002	0.409	0.0001	0.0115
Former *v.* never	0.018	0.420	0.9642	0.0000	−0.074	0.420	0.8590	0.0000
WHbR	1.898	1.288	0.1409	0.0011	1.488	1.291	0.2496	0.0006
Alcohol	—	—	—	0.0000	1.606	0.501	0.0014	0.0049

[a] The increase in the R^2 associated with the addition of each independent variable while controlling for the other independent variables.

The interaction terms for smoking and alcohol intake, and WHbR and BMI were not significant.

To illustrate the degree to which systolic blood pressure was associated with WHbR, the means for the first (lowest) and third (highest) tertiles of WHbR were used with the regression coefficients to calculate blood pressure. The mean age and BMI for the entire study group were kept constant, smoking status was defined as 'never'. Blood pressure for men with the mean WHbR for the first tertile was 122.15 mm Hg compared to 123.36 mm Hg for the third tertile. With alcohol in the model, blood pressure calculated using the mean WHbR for the first tertile was 122.09 mm Hg for men consuming less than two drinks per day and 124.75 mm Hg for men drinking two or more drinks per day. Blood pressure for men with the mean WHbR for the third tertile was 123.07 mm Hg for men consuming less than two drinks per day and 125.73 mm Hg for men consuming more than two drinks per day.

Discussion

Fat distribution was examined in relation to blood pressure in 1936 normotensive men cross-sectionally. A significant but small association was demonstrated between the ratio of waist circumference to hip breadth and systolic blood pressure after adjusting for the effects of age, cigarette smoking and BMI. Adjustment for alcohol intake attenuated the association between WHbR and blood pressure.

Our results are consistent with other studies that have demonstrated a positive association between body fat distribution and blood pressure that is independent of BMI[2,11,12,14] and age[2,10,11]. The overall amount of explained variability in blood pressure remains low even with the inclusion of all of the covariates in the model. Although the magnitude of the association we report is lower than that reported by other researchers[10–12] our results may underestimate the true strength of the association due to the health-screened nature of the cohort. In addition, few of these studies considered the influence of other known risk factors for elevated blood pressure on this association[27,34]. The body fat distribution-blood pressure relationship may be affected, for example, by smoking or the dietary intake of alcohol.

The association between alcohol and elevated blood pressure is well documented[15–22] although the strength of this relationship is unclear. MacMahon *et al.*[17] found that the mean blood pressure and the incidence of hypertension increased in those who drank more than two drinks per day and estimated that the maximum proportion of hypertension that could be attributed to alcohol was at most 7 percent. In another investigation of the Normative Aging Study cohort, using data derived from a questionnaire administered in 1982, higher mean systolic blood pressures were reported for men drinking three or more drinks per day compared to men drinking less than three drinks per day controlling for age, body weight and current smoking[22].

The effect of alcohol intake on the relationship of body fat distribution to blood pressure has not received much attention. Williams *et al.*[12] reported that percent of total dietary calories derived from alcohol was not related to blood pressure regardless of whether the correlation was adjusted for the effects of waist-to-hip ratio. Reichley *et al.*[27] found that neither waist-to-hip ratio nor alcohol when considered jointly were related to blood pressure after BMI and a mean skinfold thickness measure were controlled in the analysis.

Although alcohol may substantially contribute to total dietary calories it does not appear to affect body weight. Body mass index did not differ between men drinking less than two versus two or more drinks per day even after controlling for the effects of smoking. Our results agree in part with Camargo *et al.*[28] who found a surplus of 4000 kcal per week in those who drank four or more drinks per day but no increase in adiposity. Gruchow *et al.*[29] also reported that drinkers had a higher caloric intake, but were not more obese than nondrinkers. According to a study using Behavioral Risk Factor Survey and Health and Nutrition Examination Study II data, alcohol had only a slight effect on BMI after controlling for smoking, age, total daily caloric intake, physical activity, race, education and height[30]. Less efficient utilization of alcohol calories or interference by alcohol in the metabolism of nonalcohol calories may account for the elevated caloric intake unaccompanied by an increase in BMI[28,29,31].

Body fat distribution, as represented by the ratio of waist circumference to hip breadth, was associated with systolic blood pressure independently of age, BMI and smoking. When the model was further adjusted for alcohol intake, the relationship between body fat distribution and blood pressure was diminished and no longer statistically significant. These findings are consistent with several possible underlying relationships, but the cross-sectional nature of this study limits our ability to choose among these alternatives. Alcohol may be a confounding variable in the assessment of the relationship between waist-to-hip ratio and blood pressure. Alcohol was associated with both the ratio of waist circumference to hip breadth and with blood pressure. If alcohol is considered a confounding variable, then failure to adjust for alcohol intake would lead to a spurious relationship between waist-to-hip ratio and blood pressure. Alternatively, alcohol may alter body fat distribution and through this mechanism may act indirectly to increase blood pressure. One hypothesis consistent with this explanation is that higher levels of alcohol intake may lead to an increased accumulation of abdominal fat (as represented by the waist-to-hip ratio) independent of total adiposity which may in turn be associated with increased insulin resistance, hyperinsulinemia[31] and ultimately increased blood pressure[32]. In one of the few studies of the determinants of body fat distribution, Haffner *et al.*[33] reported that alcohol was unrelated to waist-to-hip ratio, but self-reporting may have resulted in misclassification of alcohol intake which would act to reduce its association with fat patterning. Alcohol may have both direct and indirect effects on blood pressure and may act both through waist-to-hip ratio and through other mechanisms to increase blood pressure. Alternatively, there may be unmeasured antecedent variables which explain the apparent relationships between alcohol intake, waist-to-hip ratio and elevated blood pressure. For example, a behavioral variable such as stress[34], or another dietary factor[35] besides alcohol could be related to both alcohol intake and waist-to-hip ratio as well as to blood pressure.

There are a few limitations of this investigation that should be mentioned. The measure of alcohol intake used in these analyses provided limited information on the frequency and variability of alcohol consumption. This rather restricted representation of alcohol intake may lead to misclassification of study subjects. Assuming that such misclassification is nonselective, the finding of a positive relationship between alcohol and blood pressure suggests that the true relationship may be even stronger. Nonetheless, a more precise quantification of alcohol intake would be desirable in assessing the relationship between alcohol intake, habitus and blood pressure.

Body fat distribution has been represented in epidemiologic studies by various indices consisting of either circumferences or skinfolds. In this investigation, the ratio of abdomen circumference to hip breadth was used as an index of body fat distribution instead of the more commonly used waist to hip circumference ratio. At the time this data were collected hip circumference was not a part of the anthropometric battery of measurements at the NAS. However, we do not view this as a serious limitation. Our hypothesis specifies centripetal adiposity, as represented by abdomen circumference, as the risk factor associated with increased blood pressure. The denominator of the ratio was used as a measure of peripheral adiposity to provide a comparison. Because hip breadth is a measure of hard and some soft tissue it would appear to accomplish this as well as hip circumference. Moreover, in a current sample of men from the NAS population the Pearson product-moment correlation between the ratio of waist circumference to hip breadth and the ratio of waist to hip circumference was high ($r = 0.73$).

The health screened nature of the cohort reduces the amount of variation in blood pressure. At the time the data for this investigation were collected, the population had been screened specifically for hypertension. Thus, variation in blood pressure is low in our sample. Stronger associations may only be demonstrable in samples that include a wider distribution of blood pressure.

The final limitation is the cross-sectional nature of this investigation which provides limited insight into the role played by diet, particularly alcohol, in modifying habitus and the roles of both diet and habitus in determining blood pressure. Longitudinal investigations on this and other populations are necessary to improve our understanding of these relationships.

The centripetal accumulation of body fat may be associated with physiologic changes that eventually lead to an elevated risk of cardiovascular disease[32]. It is still unclear what determines regional accumulation of body fat. Our finding of a positive relationship between cigarette smoking and WHbR in addition to alcohol agrees with that of Shimokata *et al.*[36], who demonstrated that current smokers had significantly greater waist-to-hip ratios when compared to both former and never-smokers. However, genetic factors may also be important in the determination of body fat distribution given the inconsistent relationships of body fat distribution to behavioral variables[33]. Current knowledge on this issue is scant and the extent to which body fat distribution is influenced by genetic and behavioral factors is largely unknown. Predisposed individuals, when exposed to certain environmental agents may respond by increasing their body fat. Dietary factors, such as alcohol intake are possible catalysts for centripetal fat accumulation. Casual observation reveals a propensity for alcohol users to develop a 'beer gut' or 'beer belly'. It is possible that alcohol intake may influence the relationship between centripetal fat accumulation and blood pressure.

In summary, the results of this investigation demonstrate a small but statistically significant association between body fat distribution and blood pressure independent of age and BMI. Our results also suggest that this relationship is not independent of alcohol intake and that further investigation is necessary to understand better the influence of alcohol intake on the body habitus–blood pressure relationship.

Acknowledgements—Supported by grant no. HL37871 from the National Heart, Lung and Blood Institute. Dr Cassano was supported in part by NIH Institutional Research Award no. HL07427.

References

1 Vague, J. (1956): The degree of masculine differentiation of obesities: a factor determining predisposition to diabetes, atherosclerosis, gout, and uric calculous disease. *Am. J. Clin. Nutr.* **4**, 20–34.

2 Kalkoff, R.K., Hartz, A.H. Rupley, D., Kissebah, A.H. & Kelber, S. (1983): Relationship of body fat distribution to blood pressure, carbohydrate tolerance, and plasma lipids in healthy obese women. *J. Lab. Clin. Med.* **102**, 621–627.

3 Feldman, R., Sender, A.J. & Siegelaub, A.B. (1969): Difference in diabetic and nondiabetic fat distribution patterns by skinfold measurements. *Diabetes* **18**, 478–486.

4 Hartz, A.J. Rupley, D.C. Kalkoff, R.D. & Rimm, A.A. (1983): Relationship of obesity to diabetes: influence of obesity level and body fat distribution. *Prev. Med.* **12**, 351–357.

5 Ohlson, L.-O., Larsson, B., Svardsudd, K., Welin, L., Eriksson, H., Wilhelmsen, L., Björntorp, P. & Tibblin, G. (1985): The influence of body fat distribution on the incidence of diabetes mellitus: 13.5 years of follow-up of the participants in the study of men born in 1913. *Diabetes* **34**, 1055–1058.

6 Sparrow, D., Borkan, G.A., Gerzof, S.G., Wisniewski, C. & Silbert, C.K. (1986): Relationship of fat distribution to glucose tolerance: results of computed tomography in male participants of the Normative Aging Study. *Diabetes* **35**, 411–415.

7 Freedman, D.S., Srinivasan, S.R., Burke, G.L., Shear, C.L., Smoak, C.G., Harsha, D.W., Webber, L.S. & Berenson, G.S. (1987): Relationship of body fat distribution to hyperinsulinemia in children and adolescents: the Bogalusa Heart Study. *Am. J. Clin. Nutr.* **46**, 403–410.

8 Blair, D., Habicht, J.P., Sims, E.A.H., Sylwester, D. & Abraham, S. (1984): Evidence for an increased risk for hypertension with centrally located body fat and the effect of race and sex on this risk. *Am. J. Epidemiol.* **119**, 526–540.

9 Selby, J.V., Friedman, G.D. & Quesenberry, C.P. (1989): Precursors of essential hypertension. The role of body fat distribution pattern. *Am. J. Epidemiol.* **129**, 43–53.

10 Baumgartner, R.N., Roche, A.F., Chumlea, W.C., Siervogel, R.M. & Glueck, C.J. (1987): Fatness and fat patterns: associations with plasma lipids and blood pressures in adults, 18–57 years of age. *Am. J. Epidemiol.* **126**, 614–628.

11 Gillum, R.F. (1987): The association of body fat distribution with hypertension, hypertensive heart disease, coronary heart disease, diabetes and cardiovascular risk factors in men and women aged 18–79 years. *J. Chronic Dis.* **40**, 421–428.

12 Williams, P.T., Fortmann, S.P., Terry, R.B., Garay, S.C., Vranizan, K.M., Ellsworth, N. & Wood, P.D. (1987): Associations of dietary fat, regional adiposity, and blood pressure in men. *JAMA* **257**, 3251–3256.

13 Seidell, J.C., Bakx, J.C., De Boer, E., Deurenberg, P. & Hautvast, J.G.A.J. (1985): Fat distribution of overweight persons in relation to morbidity and subjective health. *Int. J. Obesity* **9**, 363–374.

14 Larsson, B., Svardsudd, K., Welin, L., Wilhelmsen, L., Björntorp, P. & Tibblin, G. (1984): Abdominal adipose tissue distribution, obesity, and risk of cardiovascular disease and death: 13 year follow up of participants in the study of men born in 1913. *Br. Med. J.* **288**, 1401–1404.

15 Klatsky, A.L., Friedman, G.D., Siegelaub, A.B. & Gérard, M.J. (1977): Alcohol consumption blood pressure. *N. Engl. J. Med.* **296**, 1194–1200.

16 Friedman, G.D., Klatsky, A.L. & Siegelaub, A.B. (1983): Alcohol intake and hypertension. *Ann. Intern. Med.* **98**, 846–849.

17 MacMahon, S.W., Blacket, R.B., Macdonald, G.J. & Hall, W. (1984): Obesity, alcohol consumption and blood pressure in Australian men and women. The National Heart Foundation of Australia risk factor prevalence study. *J. Hypertens.* **2**, 85–91.

18 Savdie, E., Grosslight, G.M. & Adena, M.A. (1984): Relation of alcohol and cigarette consumption to blood pressure and serum creatinine levels. *J. Chronic Dis.* **37**, 617–623.

19 Puddey, I.B., Beilin, L.J., Vandongen, R., Rouse, I.L. & Rogers, P. (1985): Evidence for a direct effect of alcohol consumption on blood pressure in normotensive men. *Hypertension* **7**, 707–713.

20 Klatsky, A.L., Friedman, G.D. & Armstrong, M.A. (1986): The relationship between alcoholic beverage use and other traits to blood pressure: a new Kaiser Permanente study. *Circulation* **73**, 628–636.

21 MacMahon, S. (1987): Alcohol consumption and hypertension. *Hypertension* **9**, 111–121.

22 Glynn, R.J., Bouchard, G.R. & Hermos, J.A. (1987): Alcohol consumption, blood pressure, and aging: results from the Normative Aging Study. *Geriatric Clin. Pharmacol.* **29–45.**

23 Bell, B., Rose, C.L. & Damon, A. (1972): The normative aging study: an interdisciplinary and longitudinal study of health and aging. *Aging Hum. Dev.* **3**, 5–17.

24 Brodman, K., Erdman, A.J. & Wollf, H.G. (1956): *Cornell Medical Index Health Questionnaire.* Ithaca, NY: Cornell University Medical College.

25 Friedlaender, J.S., Costa, P.T., Bosse, R., Ellis, E., Rhoads, J.G. & Stoudt, H.W. (1977): Longitudinal physique changes among healthy white veterans at Boston. *Hum. Biol.* **49**, 541–558.

26 SAS User's Guide (1985): *Statistics, version 5 edition*, p.776. Cary, NC: Statistical Analysis System Institute, Inc.

27 Reichley, K.B., Mueller, W.H., Hanis, C.L., Joos, S.K., Tulloch, B.R., Barton, S. & Schull, W.J. (1987): Centralized obesity and cardiovascular disease risk in Mexican Americans. *Am. J. Epidemiol.* **125**, 373–386.

28 Camargo, C.A., Vranizan, K.M., Dreon, D.M., Frey-Hewitt, B. & Wood, P.D. (1987): Alcohol caloric intake, and adiposity in overweight men. *J. Am. Coll. Nutr.* **6**, 271–278.

29 Gruchow, H.W., Sobocinski, K.A., Barboriak, J.J. & Scheller, J.G. (1985): Alcohol consumption, nutrient intake and relative body weight among US adults. *Am. J. Clin. Nutr.* **42**, 289–295.

30 Williamson, D.F., Forman, M.R., Binkin, N.J., Gentry, E.M., Remington, P.L. & Trowbridge,

F.L. (1987): Alcohol and body weight in United States adults. *Am. J. Public Health* **77**, 1324–1330.

31 Shelmet, J.J., Reichard, G.A., Skutches, C.L., Hoeldtke, R.D., Owen, O.E. & Boden, G. (1988): Ethanol causes acute inhibition of carbohydrate, fat and protein oxidation and insulin resistance. *J. Clin. Invest.* **81**, 1137–1145.

32 Landsberg, L. (1986): Diet, obesity and hypertension: an hypothesis involving insulin, the sympathetic nervous system, and adaptive thermogenesis. *Q. J. Med.* **236**, 1081–1090.

33 Haffner, S.M., Stern, M.P., Hazuda, H.P., Pugh, J., Patterson, J.K. & Malina, R. (1986): Upper body and centralized adiposity in Mexican Americans and non-Hispanic whites: relationship to body mass index and other behavioral and demographic variables. *Int. J. Obesity* **10**, 493–502.

34 Björntorp, P. (1988): Abdominal obesity and the development of noninsulin-dependent diabetes mellitus. *Diabetes/Metabolism Rev.* **4**, 615–622.

35 Bouchard, C., Pérusse, L., Leblanc, C., Tremblay, A. & Thériault, G. (1988): Inheritance of the amount and distribution of human body fat. *Int. J. Obesity* **12**, 205–215.

36 Shimokata, H., Muller, D.C. & Andres, R. (1989) Studies in the distribution of body fat. III. Effects of cigarette smoking. *JAMA* **261**, 1169–1173.